Figure S1

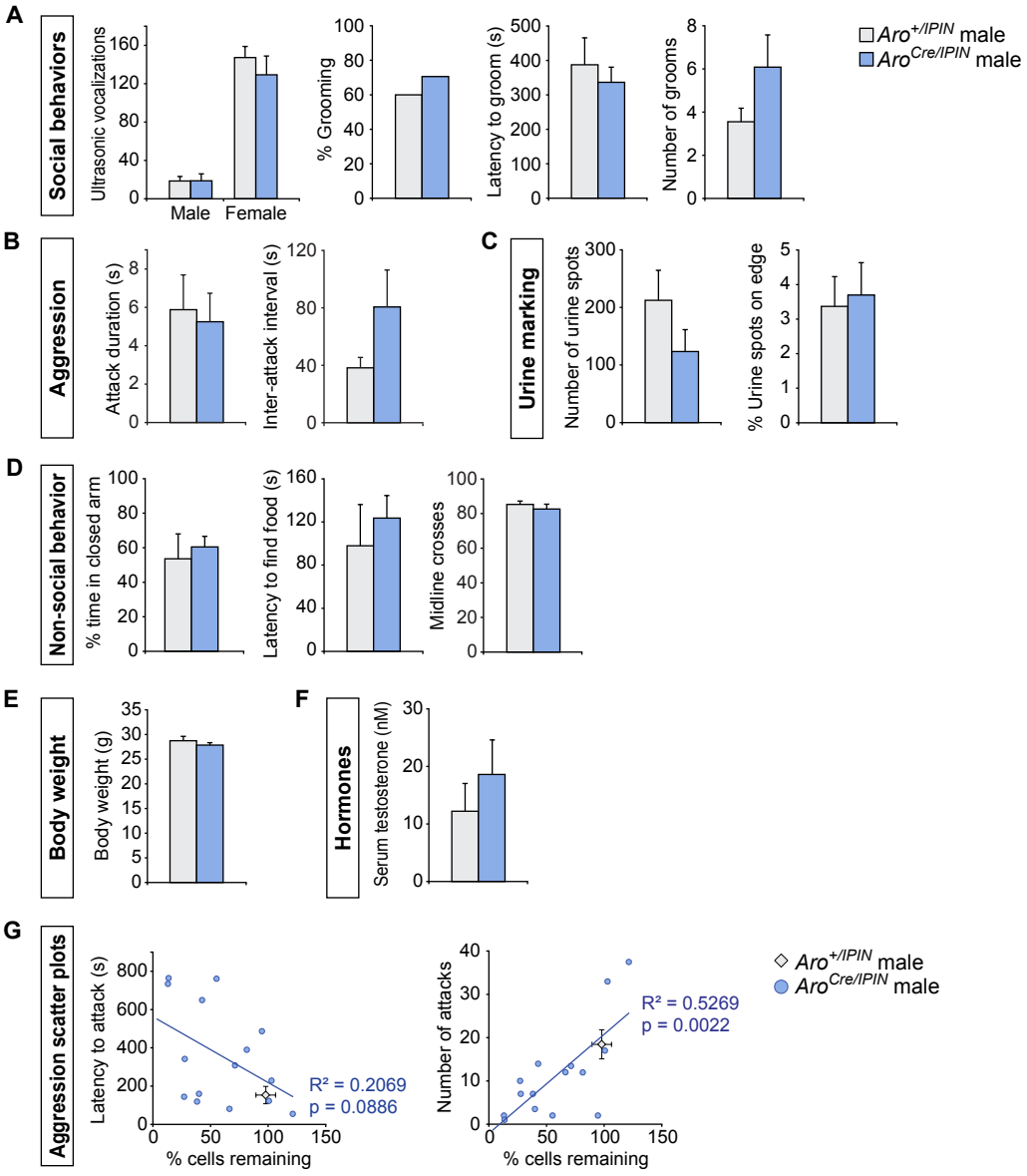
A
Social behaviors
Ultrasonic vocalizations
Male
Female
% Grooming
Latency to groom (s)
Number of grooms
Aro+/IPIN male
AroCre/IPIN male
B
Aggression
Attack duration (s)
Inter-attack interval (s)
C
Urine marking
Number of urine spots
% Urine spots on edge
D
Non-social behavior
% time in closed arm
Latency to find food (s)
Midline crosses
E
Body weight
Body weight (g)
F
Hormones
Serum testosterone (nM)
G
Aggression scatter plots
Latency to attack (s)
% cells remaining
R² = 0.2069
p = 0.0886
Number of attacks
R² = 0.5269
p = 0.0022
Aro+/IPIN male
AroCre/IPIN male

Figure S1. Ablation of male aromatase+ MeApd neurons reduces specific components of aggression, Related to Figure 2.

(A) No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ males in emitting ultrasonic vocalizations toward WT male and female intruders or in grooming WT intruder males.

(B) No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ males in pattern of attacks (mean attack bout duration and inter-attack interval) toward a WT intruder male.

(C) No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ males in marking territory with urine.

(D) No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ males in anxiety-type behavior (elevated-plus maze), finding food when starved, or locomotor activity as measured by number of midline crosses.

(E, F) No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ males in body weight or serum testosterone.

(G) Significant correlation between loss of aromatase+ MeApd neurons and number of attacks toward a WT intruder male. Scatter plots include data from all $aro^{Cre/IPIN}$ males who displayed any attacks (15/27); gray diamond in the plots depicts number of attacks by $aro^{+/IPIN}$ males toward WT intruder males.

Mean ± SEM; n = 14 $aro^{+/IPIN}$ (A-G), n = 15 $aro^{Cre/IPIN}$ (A-F), n = 27 $aro^{Cre/IPIN}$ (G).

Figure S2

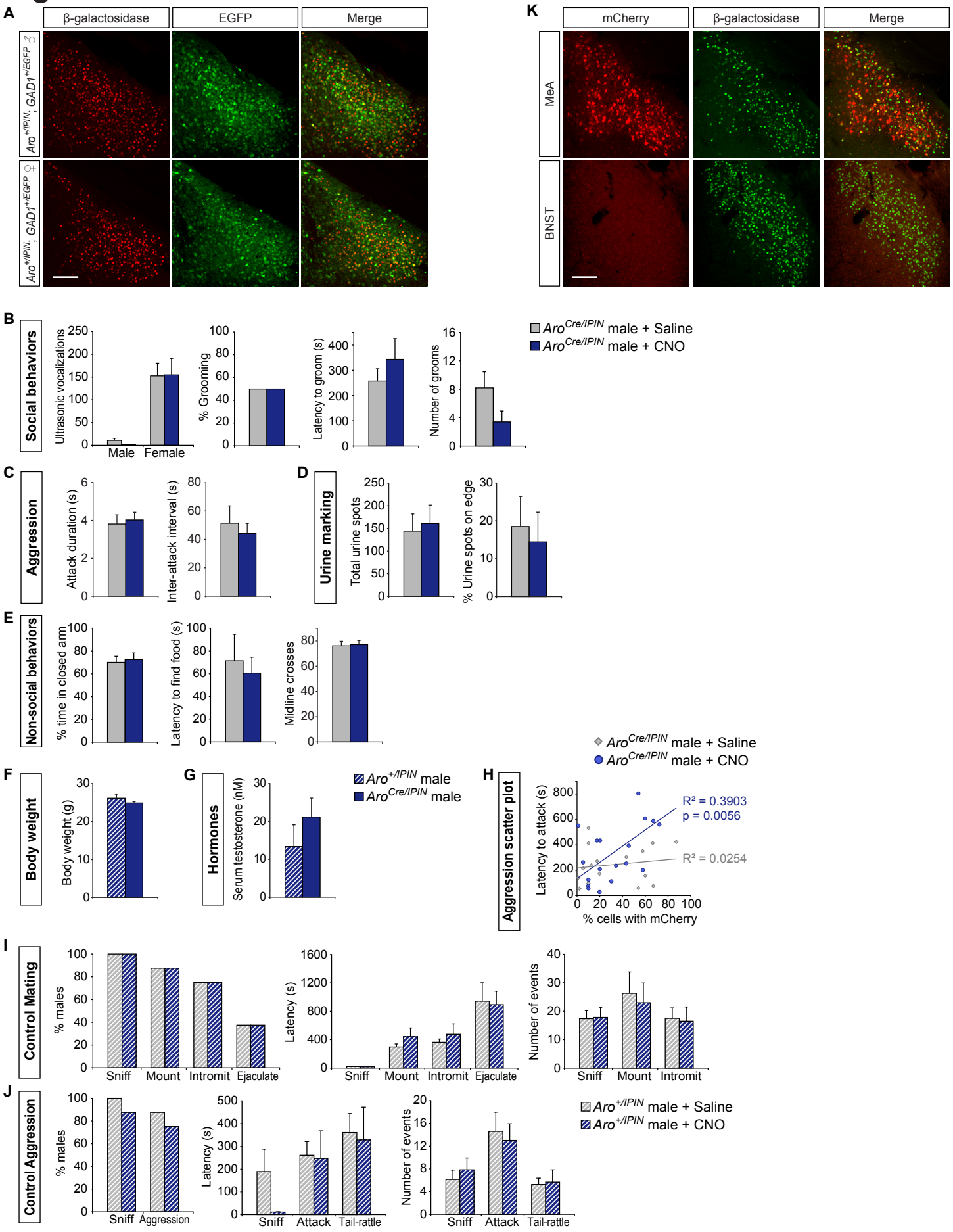
A
β-galactosidase
EGFP
Merge
Aro+/IPIN; GAD1+/EGFP ♂
Aro+/IPIN; GAD1+/EGFP ♀
K
mCherry
β-galactosidase
Merge
MeA
BNST
B
Social behaviors
Ultrasonic vocalizations
Male
Female
% Grooming
Latency to groom (s)
Number of grooms
AroCre/IPIN male + Saline
AroCre/IPIN male + CNO
C
Aggression
Attack duration (s)
Inter-attack interval (s)
D
Urine marking
Total urine spots
% Urine spots on edge
E
Non-social behaviors
% time in closed arm
Latency to find food (s)
Midline crosses
F
Body weight
Body weight (g)
G
Hormones
Serum testosterone (nM)
Aro+/IPIN male
AroCre/IPIN male
H
Aggression scatter plot
AroCre/IPIN male + Saline
AroCre/IPIN male + CNO
Latency to attack (s)
% cells with mCherry
R² = 0.3903
p = 0.0056
R² = 0.0254
I
Control Mating
% males
Latency (s)
Number of events
Sniff
Mount
Intromit
Ejaculate
J
Control Aggression
% males
Latency (s)
Number of events
Sniff
Aggression
Attack
Tail-rattle
Aro+/IPIN male + Saline
Aro+/IPIN male + CNO

Figure S2. Inhibiting male aromatase+ MeApd neurons reduces specific components of aggression, Related to Figure 3.

(A) Most aromatase+ MeApd neurons co-express GAD1, a biosynthetic enzyme for the neurotransmitter GABA. (% aromatase+ MeApd neurons that are GAD1+: Male, 83.7% ± 3.4; Female, 90.6% ±4.8; n = 2 for each sex).

(B) No difference between $aro^{Cre/IPIN}$ males given CNO or saline in emitting ultrasonic vocalizations toward WT male and female intruders or in grooming WT intruder males.

(C) No difference between $aro^{Cre/IPIN}$ males given CNO or saline in pattern of attacks (mean attack bout duration and inter-attack interval) toward a WT intruder male.

(D) No difference between $aro^{Cre/IPIN}$ males given CNO or saline in marking territory with urine.

(E) No difference between $aro^{Cre/IPIN}$ males given CNO or saline in anxiety-type behavior (elevated-plus maze), finding food when starved, or locomotor activity as measured by number of midline crosses.

(F, G) No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ males in body weight or serum testosterone.

(H) Significant correlation between more aromatase+ MeApd neurons expressing DREADD-G_i and increased latency to attack WT intruder male following CNO administration. Scatter plot includes data from all $aro^{Cre/IPIN}$ males who displayed any attacks (18/28, CNO; 16/28, saline).

(I, J) No difference between $aro^{+/IPIN}$ males given CNO or saline in mating or aggression with WT female and male intruders, respectively.

(K) Injection of AAV encoding the Cre-dependent fusion protein DREADD-G_i:mCherry into the MeA results in expression of mCherry in this region but not in other aromatase+ (Cre+) regions such as the BNST.

Mean ± SEM; n = 8 $aro^{+/IPIN}$ (F, G, I, J), n = 20 $aro^{Cre/IPIN}$ (B-G), n = 28 $aro^{Cre/IPIN}$ (H).

Scale bars = 200 μm.

Figure S3

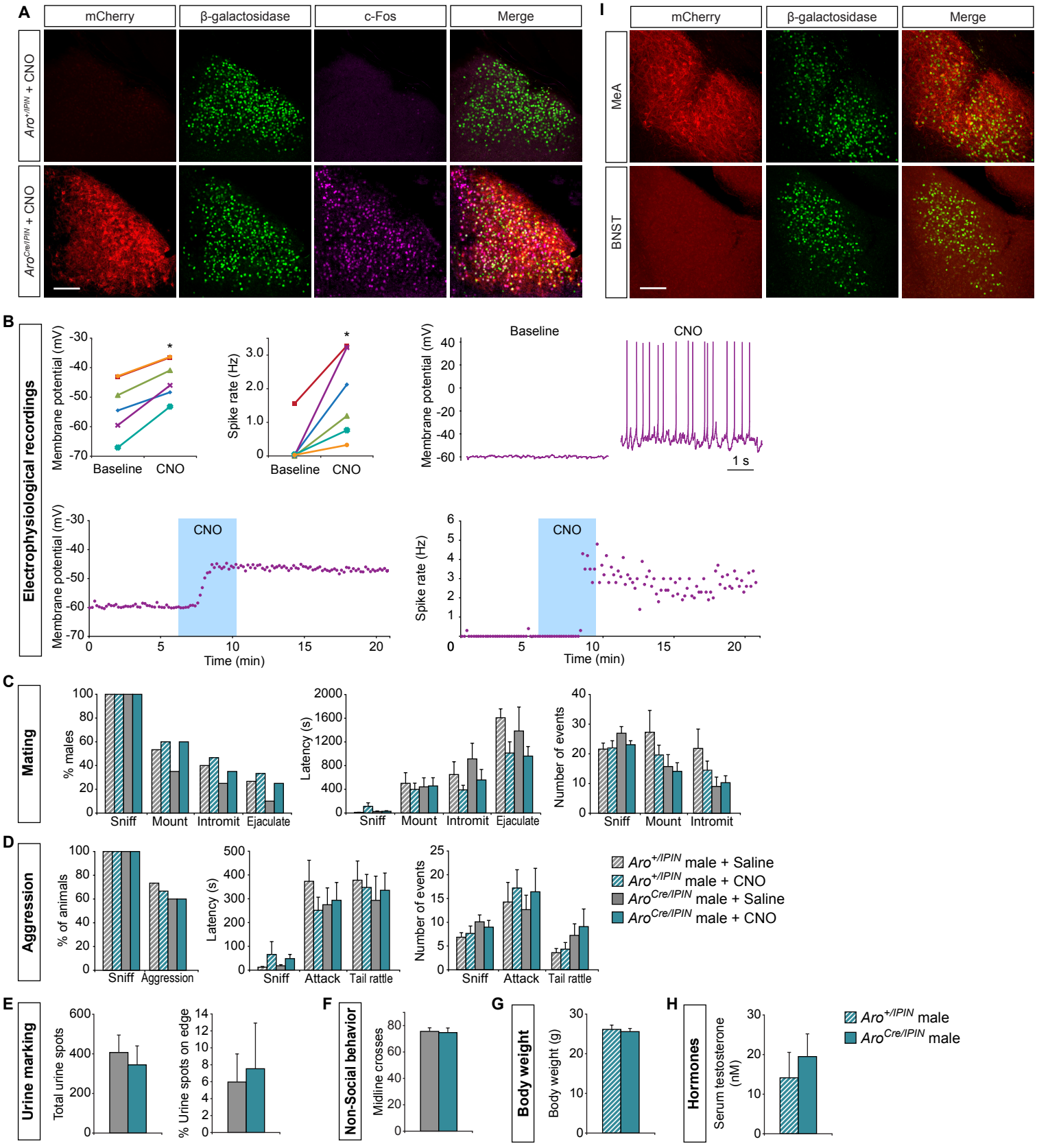
A
mCherry
β-galactosidase
c-Fos
Merge
Aro+/IPIN + CNO
AroCre/IPIN + CNO
I
mCherry
β-galactosidase
Merge
MeA
BNST
B
Electrophysiological recordings
Membrane potential (mV)
Baseline
CNO
Spike rate (Hz)
1 s
Time (min)
C
Mating
% males
Latency (s)
Number of events
Sniff
Mount
Intromit
Ejaculate
D
Aggression
% of animals
Attack
Tail rattle
Aro+/IPIN male + Saline
Aro+/IPIN male + CNO
AroCre/IPIN male + Saline
AroCre/IPIN male + CNO
E
Urine marking
Total urine spots
% Urine spots on edge
F
Non-Social behavior
Midline crosses
G
Body weight
Body weight (g)
H
Hormones
Serum testosterone (nM)
Aro+/IPIN male
AroCre/IPIN male

Figure S3. Activating male aromatase+ MeApd neurons does not modulate behavior, Related to Figure 3.

AAV encoding Cre-dependent DREADD-G_q:mCherry was injected bilaterally into the MeA of *aro*$^{Cre/IPIN}$ and *aro*$^{+/IPIN}$ males, and animals were allowed to recover for >10 days.

(A) CNO administration leads to c-Fos activation in MeApd of *aro*$^{Cre/IPIN}$ but not *aro*$^{+/IPIN}$ males.

(B) Electrophysiological recordings were performed on coronal brain slices (225-230 μm thick) containing the MeA. Aromatase+ neurons expressing DREADD-G_q were selected for patch-clamp recording based on mCherry expression. Both membrane potential and spike rate increased after the application of 1 μM CNO in aCSF for 4 min. Panels at top left show increases in membrane potential and spike rate in the presence of CNO for each of the 6 neurons (each cell represented in a different color). Other panels show changes in membrane potential and spike rate in an example neuron; the blue box denotes time of CNO application. n = 6 cells from 4 animals. *$p < 0.05$ (sign-rank test).

(C-F) No difference between *aro*$^{Cre/IPIN}$ and *aro*$^{+/IPIN}$ males given saline or CNO in displays of mating, aggression, urine marking, or locomotor behavior.

(G, H) No difference between *aro*$^{Cre/IPIN}$ and *aro*$^{+/IPIN}$ males in body weight or serum testosterone.

(I) Injection of AAV encoding the Cre-dependent fusion protein DREADD-G_q:mCherry into the MeA results in expression of mCherry in this region but not in other aromatase+ (Cre+) regions such as the BNST.

Mean ± SEM; $n \geq 5$ for both genotypes (A), n = 5 *aro*$^{+/IPIN}$ (B-G), n = 24 *aro*$^{Cre/IPIN}$ (B-G).

Scale bars = 200 μm.

Figure S4

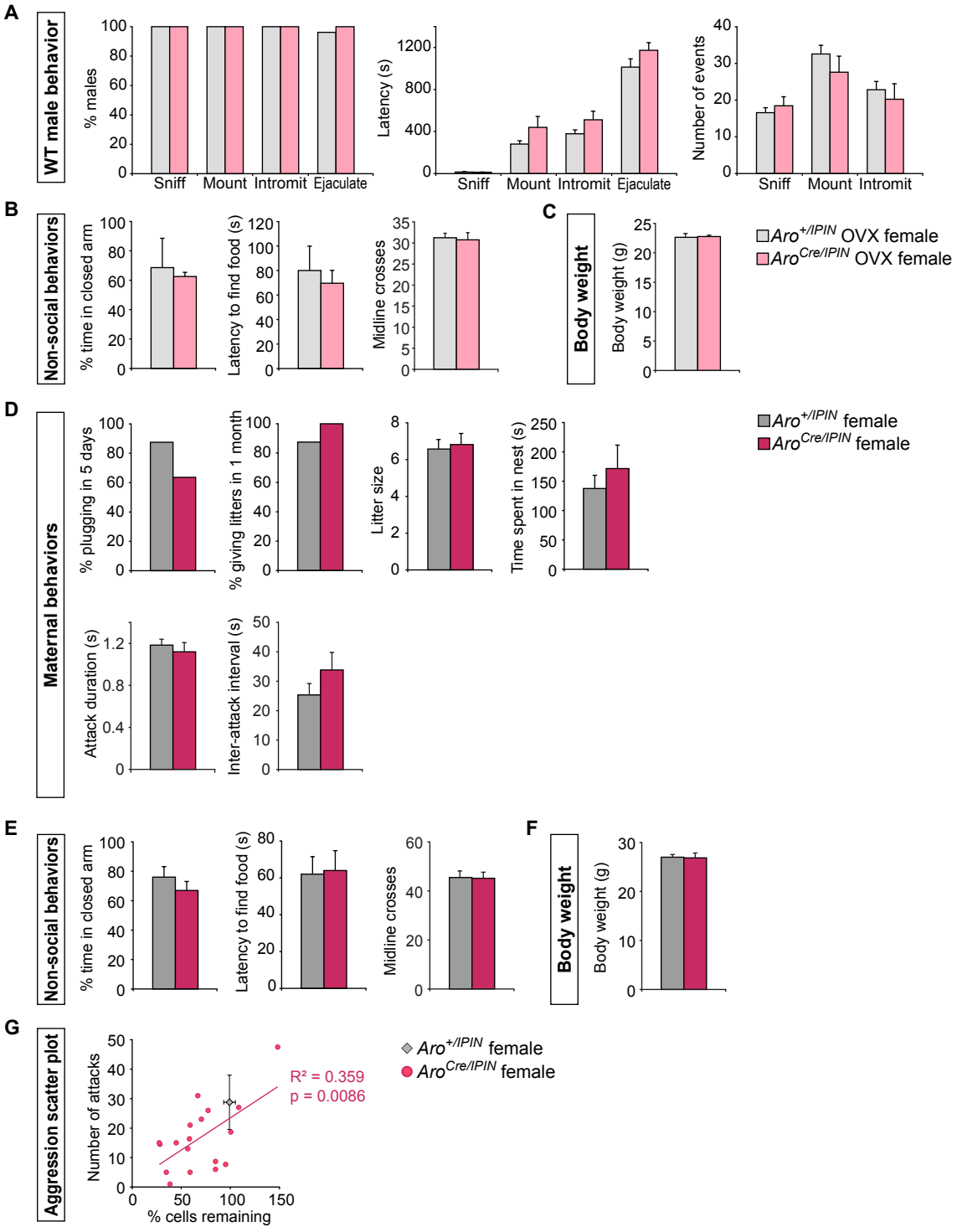
A
WT male behavior
% males
Sniff
Mount
Intromit
Ejaculate
Latency (s)
Number of events
B
Non-social behaviors
% time in closed arm
Latency to find food (s)
Midline crosses
C
Body weight
Body weight (g)
Aro+/IPIN OVX female
AroCre/IPIN OVX female
D
Maternal behaviors
% plugging in 5 days
% giving litters in 1 month
Litter size
Time spent in nest (s)
Attack duration (s)
Inter-attack interval (s)
Aro+/IPIN female
AroCre/IPIN female
E
Non-social behaviors
% time in closed arm
Latency to find food (s)
Midline crosses
F
Body weight
Body weight (g)
G
Aggression scatter plot
Number of attacks
% cells remaining
R² = 0.359
p = 0.0086
Aro+/IPIN female
AroCre/IPIN female

Figure S4. Ablation of aromatase+ MeApd neurons reduces specific components of maternal aggression, Related to Figure 4.

For tests of sexual behavior (A-C), $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ females were ovariectomized (OVX) and estrus was hormonally induced prior to testing. For tests of maternal behavior (D-G), $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ females were gonadally intact, mated with a WT male, and allowed to deliver a litter.

(A) WT males mate equivalently with $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ females.

(B) No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ females in anxiety-type behavior (elevated-plus maze), finding food when starved, or locomotor activity as measured by number of midline crosses.

(C) No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ females in body weight.

(D) No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ females in percent plugged by WT male, percent producing litters, litter size, and time spent in the nest in 15 min during pup retrieval. No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ mothers in pattern of attacks (mean attack bout duration and inter-attack interval) toward a WT intruder male.

(E) No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ females in anxiety-type behavior (elevated-plus maze), finding food when starved, or locomotor activity as measured by number of midline crosses.

(F) No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ females in body weight.

(G) Significant correlation between extent of loss of aromatase+ MeApd neurons and reduced number of attacks toward WT intruder male. Scatter plot includes data from all $aro^{Cre/IPIN}$ mothers who displayed any attacks; gray diamond in the plot depicts number of attacks by $aro^{+/IPIN}$ mothers toward WT intruder males (18/28).

Mean ± SEM; n ≥ 14 $aro^{+/IPIN}$ (A-G), n = 7 $aro^{Cre/IPIN}$ (A-C), n = 8 $aro^{Cre/IPIN}$ (D-F), n = 28 $aro^{Cre/IPIN}$ (G)

Supplemental Movie Legends

Movie S1: Resident male attacks intruder male, Related to Figure 4.

A WT resident male is shown attacking a WT intruder male with attacks to the flank, wrestling, and boxing.

Movie S2: Maternal aggression toward an intruder male, Related to Figure 4.

A WT mother (pups have been removed for safety) shows aggression toward a WT intruder male by directing attack toward the genital area.

Supplemental Experimental Procedures

Animals

Adult mice 10–24 weeks of age were used in all studies. Mice were housed in a UCSF barrier facility with a 12:12 hr light:dark cycle, and food and water were available *ad libitum*. *Aro*$^{Cre/IPIN}$ mice and their control *aro*$^{+/IPIN}$ same-sex siblings were used for behavioral studies. In order to examine GABA co-localization with aromatase, we mated *aro*$^{+/IPIN}$ mice with mice bearing an EGFP knocked-in to the *GAD1* locus such that it faithfully colabels GABA neurons within the MeA and examined progeny doubly heterozygous for *LacZ* and *egfp* (Bian, 2013; Tamamaki et al., 2003). Animals were group-housed by sex after weaning at 3 weeks of age. All studies with animals were done in accordance with UCSF IACUC protocols.

Generation of *aromatase*^{Cre} mice

We used previously described homology DNA arms to generate a targeting construct for the *aromatase* locus (Wu et al., 2009). A transgene encoding IRES-Cre recombinase followed immediately by a previously described FRT-flanked neomycin selection (FNF) cassette was inserted as an *AscI* fragment 3 bp 3' of the stop codon in the last exon of the *aromatase* locus in mouse 129/SvEv ES cells (Yang et al., 2013). We screened G418-resistant ES clones for homologous recombination of the 3' arm with PCR, using a primer (5'-CATCGCCTTCTATCGCCTTCTTGAC, F2) that was complementary to the neomycin selection cassette and an external primer (5'-CTTGATCATTGGAGCCAAATCTGGATG, R2) that was complementary to genomic sequence located 3' of the 3' homology arm of the targeting vector. A subset of positive clones was tested by PCR for homologous targeting of the 5' arm using an external primer (5'-CCAGCTGGATTTCTTGGGATCAAATTCAGG, F1) and a primer unique

to the modified allele (5'-GAATTCGGCGCGCCTCTTCACTGTTG, R1). ES clones harboring the homologously recombined modified aromatase allele were injected into blastocysts to obtain chimeric mice that were crossed to C57Bl/6J females to obtain germline transmission. Chimeric mice that transmitted the *aro*^{Cre} allele were obtained from one positive clone. F1 progeny of these chimeric mice were mated with mice expressing Flpe recombinase ubiquitously to delete the FNF cassette (Rodríguez et al., 2000). This deletion event was verified with PCR, using a primer complementary to Cre recombinase (5'-AGGATGACTCTGGTCAGAGATACCTG, F3) and the external primer R2. Progeny with a successful deletion of this selection cassette were backcrossed >5 generations in C57Bl/6J and used for experimental analysis.

Stereotaxic surgery and viruses

Stereotaxic surgery was performed as described previously (Morgan et al., 2014; Yang et al., 2013). Briefly, a mouse was placed in a stereotaxic frame (Kopf Instruments) under anesthesia (0.5-2% isofluorane), the skull was exposed with a midline scalp incision, and the stereotaxic frame was aligned at bregma using visual landmarks. The drill (drill bit #85; ~279 µm diameter) on the stereotaxic frame was placed over the skull at coordinates corresponding to the MeA (rostrocaudal, −1.6 mm; mediolateral, ± 2.2 mm), and a hole was drilled through the skull bone to expose the brain. A 33 gauge steel needle connected via PE20 tubing to a Hamilton syringe was loaded with virus, aligned at bregma (including in the z-axis) and slowly lowered to a depth of 5.15 mm. Virus was delivered bilaterally (1 µl of the previously described AAV-flex-taCasp3-TEVp (Yang et al., 2013), or 0.7 µl of AAV-flex-DREADD-G_i:mCherry, or 0.7 µL of AAV-flex-DREADD-G_q:mCherry) at 100 nL/min with a Hamilton syringe using a micropump (Harvard Apparatus). We used AAV serotypes 1 or 10 for delivering caspase-3 and AAV

serotype 10 for all DREADD studies. AAV serotype 1 virus (3 x 10^{12} IU/mL) was generated at the University of North Carolina Vector core, and AAV serotype 10 viruses (1 x 10^{13} genomes copies/mL) were packaged by PMF and colleagues. The needle was left in place for an additional 5 min to allow diffusion of the virus and then was slowly withdrawn. Mice used for receptivity assays were ovariectomized during stereotaxic surgery while the virus was being delivered. Mice were weighed and allowed to recover on a heating pad before being returned to their home cage.

Behavior

Behavioral testing was performed ≥ 1 hr after onset of the dark cycle and recorded and analyzed as described previously, except for testing in the elevated-plus maze which was performed 2-3 hours before the onset of the dark cycle (Wu et al., 2009; Yang et al., 2013). There were ≥ 2 days between behavioral tests, and residents were always exposed to a novel intruder. For sexual receptivity assays, sequential tests were 7 days apart to allow hormone levels to subside prior to estrus induction for the next assay.

Male residents were singly housed 7-10 days prior to behavior testing. Briefly, they were tested twice for mating (30 min with an estrus female), once for territory marking (1 hr in a fresh cage lined with Whatman filter paper), twice for aggression (15 min with a group housed intruder male), and once for ultrasonic vocalization (3 min with a male intruder, then 3 min with female intruder). For studies with DREADDs, CNO and saline were administered randomly such that 50% of the mice received CNO (or saline) on the first trial, and the treatment was reversed on the second trial; in these studies, males were tested once each with CNO and saline in tests of mating, territory marking, aggression, ultrasonic vocalization, and performance on an

elevated-plus maze (as described below). Animals were tested 30 min subsequent to intraperitoneal injection of CNO or saline.

For tests of sexual receptivity, a group-housed ovariectomized female was hormonally primed to be in estrus (see below) and inserted for 30 min into the cage of a sexually experienced WT resident male. The female was returned to group-housing following such an assay. All females were tested three times for sexual receptivity. For tests of maternal behavior, females were allowed one week to recover from stereotaxic surgery and then co-housed with a sexually experienced male. These females were checked daily for the presence of vaginal plugs for the first 5 days. They were singly housed 3-5 days prior to parturition and checked daily for the presence of a litter. Upon parturition the cage was examined for the presence of placentae and to determine whether pups had been licked clean and gathered into nest. Nursing females were tested for pup retrieval twice for 15 min each with 2-6 day old pups. Four pups from the nest were scattered across the cage, and various parameters of pup retrieval were scored. Females were tested twice for maternal aggression 6-10 days after parturition. The pups were removed and a group-housed WT intruder male was inserted into the nursing female's cage for 15 min. The pups were returned to the dam at the end of the assay.

Once all tests of social behavior were completed, males and females were tested once on an elevated-plus maze for 5 min after being placed in the center, open compartment. They were subsequently tested for 5 min for their ability to find a cracker (Cheez-It or Goldfish) after being deprived of food for 6 hr. Following behavior testing, mice were weighed, blood was collected for hormone titers, and they were perfused for histological analysis. All tests were scored by an

experimenter blind to the genotype and drug treatment of the mice, using a software package we developed in MATLAB (Wu et al., 2009).

Histology

Animals were perfused with 4% paraformaldehyde as described previously (Yang et al., 2013), and sections were collected at a thickness of 65 μm for immunolabeling or 100 μm for *in situ* hybridization using a vibrating microtome (Leica). Immunolabeling was performed using previously published protocols (Wu et al., 2009). *In situ* hybridization was performed as described previously, using published probes for aromatase and Cre recombinase (Wu et al., 2009; Yang et al., 2013). The primary antisera used are: chicken anti-β-gal (Abcam, 1:6,000), rabbit anti-GFP (Invitrogen, 1:2,000), rabbit anti-c-Fos (Millipore, 1:10,000), rat anti-dsRed (Clontech, 1:2,000), and alkaline phosphatase-conjugated sheep anti-digoxygenin (Roche, 1:5,000). The fluorophore conjugated secondary antisera are: Cy3 donkey anti-rabbit, Cy3 donkey anti-rat, Cy3 donkey anti-chicken (Jackson ImmunoResearch, 1:800), AlexaFluor 647 donkey anti-chicken (Jackson ImmunoResearch, 1:500), and AlexaFluor 488 donkey anti-rabbit, AlexaFluor 488 donkey anti-chicken (Invitrogen, 1:300).

Sections were imaged using a confocal microscope (Zeiss). To estimate cell loss following caspase-3 mediated ablation, images were obtained from every section spanning the MeApd, using a 20X objective to collect z-stacks with a 2 μm step. These images were processed in Fiji software prior to enumeration. We improved signal:noise by subtracting any auto-fluorescence from the imaging channel as necessary and applying a Gaussian blur protocol. We subsequently used the plug-in Image-based Tool for Counting Nuclei (Byun et al., 2006). In initial studies, these automated count estimates were validated with manual counts of cell

number in randomly chosen sections through the MeApd; we also routinely performed manual enumeration in randomly chosen sections to validate automated estimates of cell counts for every experiment. All experimental and control animals for a given experiment were analyzed using the same set of parameters in Fiji to minimize any bias. Only animals where both the left and right MeApd showed cell loss of >50% compared to the number of aromatase neurons in the control group were included in the analysis shown in bar graph plots. The scatterplot data were plotted using cell numbers from the side of the MeApd (left or right) with the most cells surviving (least efficient cell death). A Pearson correlation coefficient was used to determine the relation between the behavioral deficit and cell loss. In studies that sought to determine overlap of markers (GAD1, mCherry) with nuclear β-galactosidase (*aro*^{IPIN}), histological sections were obtained, imaged, and initially processed in Fiji as described above. β-galactosidase+ nuclei were identified, and the average pixel intensity signal reflecting labeling for GAD1 or mCherry was measured in the appropriate channel. β-galactosidase+ nuclei co-labeled with a minimum average pixel intensity exceeding noise in the relevant channel were deemed to co-express GAD1 or mCherry as appropriate.

Hormones and Drugs

Hormone titers were determined from blood drawn via cardiac puncture prior to perfusion or submandibular vein puncture, using kits and protocols from DRG International. Estrus was induced in ovariectomized females as described previously (Yang et al., 2013). Briefly, 17 β-estradiol was injected subcutaneously on days -2 and -1 (10 μg in 100 μL sesame oil and 5 μg in 50 μL sesame oil, respectively), and progesterone was injected subcutaneously on day 0 (50 μg in 50 μL sesame oil) 4-6 hours prior to behavioral testing.

CNO dissolved in saline (0.5 mg/mL) was injected intraperitoneally to achieve a dose (5 mg/kg) consistent with previous studies (Ray et al., 2011; Sasaki et al., 2011), and control animals received an equivalent volume of saline.

Electrophysiology

As described above, AAV-flex-DREADD-G_q:mCherry was injected bilaterally into the MeA of *aro*$^{IPIN/Cre}$ males 8 weeks of age. The mice were allowed to recover >10 days from surgery and then sacrificed brain slice preparation. Slices were cut in ice-cold HEPES buffer solution, incubated for 15 min at 33 ºC in NMDG recovery solution, and finally maintained for 1-5 hrs before recording at room temperature in HEPES buffer solution. HEPES solution composition (in mM): 92 NaCl, 2.5 KCl, 1.2 NaH_2PO_4, 30 $NaHCO_3$, 20 HEPES, 25 Glucose, 5 Na^+ Ascorbate, 2 Thiourea, 3 Na^+ Pyruvate, 10 $MgSO_4$, 0.5 $CaCl_2$, 305 mOsm, 7.3-7.4 pH. NMDG solution was identical to HEPES, except for an equimolar replacement of NaCl for NMDG.

Neurons were visualized under differential interference contrast optics and epifluorescence and selected for recording based on mCherry expression. Whole cell current-clamp recordings were made using the following recording solutions (in mM): *Internal*: 9 HEPES, 113 K-Gluconate, 4.5 $MgCl_2$, 0.1 EGTA, 14 Tris-phosphocreatine, 4 Na_2ATP, 0.3 Tris-GTP, 10 Sucrose, 290 mOsm, 7.2-7.25 pH ; *External*: 125 NaCl, 2.5 KCl, 1 $MgCl_2$, 1.25 NaH_2PO_4, 25 $NaHCO_3$, 25 Glucose, 2 $CaCl_2$, 305 mOsm, 7.25-7.30 pH. Synaptic transmission was maintained to allow for any potential DREADD effects on synaptic input. Spontaneously spiking cells (n = 4 of 6 cells) were held with constant negative holding current ("bias current", -

18.9 ± 7.2 pA) to prevent cells from entering depolarization block with CNO application. Bias current was determined before the baseline period and was held constant throughout individual recordings. V_m were not corrected for junction potential (12 mV). All statistics were performed in MATLAB and exclusion criteria were as follows: if cells did not appear to have a stable baseline during the experiment, CNO was not applied. Linear regression was also performed on the baseline period V_m for each cell; if a statistically significant ($p < 0.05$) slope of greater than ± 0.5 mV/min was observed during this period, the cell was excluded from analysis ($n = 0$ cells).

Data Analysis

Behavioral and histological studies were performed and analyzed by an experimenter blinded to the genotype and to other experimental manipulations (such as CNO versus saline treatment in DREADD studies). For analysis of non-categorical parameters of mating, aggression, receptivity and maternal care, we only included data from the animals that performed the behavior. In instances where an animal was tested more than once in the same assay (male and female mating, intermale and maternal aggression, and pup retrieval subsequent to caspase-3 mediated ablation) the behavioral performance for each animal was averaged prior to further analysis across animals. Linear regression analysis was performed using MATLAB and R. We used Fisher's exact test to analyze categorical data. For all other comparisons, we first analyzed the data distribution with the Lilliefors' goodness-of-fit test of normality. Data sets not violating this test of normality were analyzed with Student's *t* test; otherwise we used the nonparametric Wilcoxon rank sum test.

Supplemental References

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